Severe aortic regurgitation due to endocarditis in a horse

Erge aortaklepinsufficiëntie ten gevolge van endocarditis bij een paard

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ABSTRACT

A 4-year-old Belgian Warmblood mare was presented because of fever, exercise intolerance, a loud diastolic cardiac murmur and a remarkable bounding pulsation that was palpable all over the body. This bounding pulsation appeared simultaneous with the cardiac contractions. Cardiac ultrasound revealed a vegetation on the aortic valve with severe aortic regurgitation and a second vegetation at the sinus of Valsalva. A reverse flow in the common carotid artery was present during diastole. Left heart catheterization showed left ventricular and aortic pressure curves characteristic of a rather acute development of the lesion. The strong bounding pulsation was caused by severe aortic regurgitation that resulted in a very wide pulse pressure with the occurrence of "Watson's water hammer pulse". Due to the grave prognosis, treatment was not attempted. Necropsy confirmed aortic valve endocarditis.

SAMENVATTING

Een 4 jaar oude Belgische warmbloedmerrie werd aangeboden wegens koorts, inspanningsintolerantie, een luid diastolisch bijgeruis op het hart en een uitgesproken kloppende slag die over het hele lichaam te voelen was. Deze pulsatie trad simultaan op met de hartslag. Door middel van echocardiografie werd een vegetatief letsel ter hoogte van de aortakleppen vastgesteld met een erge klepinsufficiëntie en een bijkomende massa ter hoogte van de sinus van Valsalva van de aorta. In de *arteria carotis communis* werd tijdens de diastole een omgekeerde flow gemeten. De drukpatronen in het linkerventrikel en de aorta wezen op een recentelijk ontstaan probleem. De uitgesproken kloppende slag kon worden verklaard door de erge aortaklepinsufficiëntie die aanleiding gaf tot een zogenaamde Watson's waterhamerpols. Wegens de zeer slechte prognose werd besloten om geen behandeling in te stellen en het paard te laten inslapen. De endocarditis van de aortakleppen werd bevestigd bij autopsie.

INTRODUCTION

Bacterial endocarditis, also known as vegetative or infective endocarditis, refers to a hematogenous infection within the heart. Infective endocarditis usually involves bacteria, though fungi, parasites and even viruses have also been reported as causative agents. *Streptococcus* spp. and *Actinobacillus* spp. are the most common isolates in equine endocarditis, although a wide range of pathogens has been reported. The strain of bacteria isolated likely depends on the environment, the cause of the infection and the type of antibiotics already given to the patient (Buergelt *et al.*, 1985; Maxson and Reef, 1997; Bonagura and Reef, 2004; Jesty and Reef, 2006).

Endocarditis is frequently diagnosed in other domestic animal species and in humans, but it is rare in horses (Buergelt *et al.*, 1985; Reef and Spencer, 1987). Young animals are more frequently affected. There is no breed predisposition (Buergelt *et al.*, 1985; Maxson and Reef, 1997; Porter *et al.*, 2008). Although a higher predisposition for males has been reported in humans and dogs, no gender predilection seems to be present in horses (Jesty and Reef, 2006; Maxson and Reef, 1997; Porter *et al.*, 2008).

Some strains of bacteria, such as *Staphylococcus aureus* and various streptococcal species, do not require damaged endothelial tissue for their adherence (Verdegaal and Sloet van Oldruitenborgh-Oosterbaan, 2006). However, other strains of bacteria require predisposing factors for their colonization, such as platelet aggregation and high circulating agglutinins, or microscopic endocardial tears or lesions (Jesty and Reef, 2006). Therefore, a higher prevalence of endocarditis on the left side of the heart is observed, probably due to increased endocardial lesions induced by the higher pressure at this site. As such, the mitral valve and the aortic valve are most commonly affected. Tricuspid valve lesions have a lower prevalence and the pulmonary valve is rarely affected. Mural endocarditis has already been described in the horse, but is the least likely to occur (Buergelt *et al.*, 1985; Reef and Spencer, 1987; Max-

son and Reef, 1997; Bonagura and Reef, 2004). Although jugular vein thrombophlebitis (Maxson and Reef, 1997) and tooth extraction (Verdegaal *et al.*, 2006) have been identified as the cause of tricuspid valve endocarditis, most frequently the exact cause of the endocarditis is unknown (Verdegaal and Sloet van Oldruitenborgh-Oosterbaan, 2006).

Horses with endocarditis are generally referred because of fever, tachycardia and a cardiac murmur (Maxson and Reef, 1997). Other clinical signs include shifting lameness, intermittent joint distension, tachypnea/dyspnea, coughing, anorexia or inappetence, weight loss, depression, arrhythmia, septic jugular vein thrombosis, edema of the head, diarrhea, colic, seizure, umbilical infection and laminitis (Maxson and Reef, 1997; Porter *et al.*, 2008). A recent study showed that hyperthermia, synovial distension and lameness are signs that significantly increase the likelihood of endocarditis (Porter *et al.,* 2008). This demonstrates that endocarditis is a systemic illness that follows a state of transient or persistent bacteremia and exposure of the endocardium to the infective agents (Verdegaal and Sloet van Oldruitenborgh-Oosterbaan, 2006).

CASE DETAILS

A 4-year-old Belgian Warmblood mare (435 kg, 155 cm height at the withers) was referred with a history of weight loss in the preceding 4 weeks and fever with exercise intolerance in the preceding 2 weeks. Despite antibiotic treatment, the fever persisted. Over the past days, the mare had become dull and showed anorexia. The animal was then referred to the clinic because of a cardiac murmur and a bounding pulsation that was palpable all over the body.

On admission, the mare was depressed and cachectic, and it had a rough hair coat. Ventral edema of the abdomen and distal edema on the four limbs were present. The fetlock joints were mildly distended but there was no lameness. The rectal temperature was 38.7ºC and the heart rate was 64 beats/min. The mucous membranes, capillary refill time and jugular vein filling were normal. Palpation of the facial artery revealed a bounding pulsation. All over the body surface, palpation with the flat hand revealed a remarkable bounding pulsation that appeared simultaneous with the cardiac contractions. The respiratory rate was 44 breaths/min with an increased respiratory effort.

Cardiac auscultation from the left side revealed a grade 5/6 holodiastolic, decrescendo murmur over the aortic valve area that ended just before the first heart sound, and a 1/6 ejection type murmur also over the aortic valve area. On the right side, a 4/6 holodiastolic murmur and a 1/6 ejection type murmur were heard over the heart base. Lung auscultation revealed crackles.

Biochemistry did not demonstrate abnormal liver, kidney or muscle function. The mare presented a total white blood cell count of 19600 cells/µL (ref. 5400-14300 cells/µL) (Morris, 2009), with 17000 cells/µL (87%) neutrophils (ref. 2300-8600 cells/µL) (Morris, 2009) and 2500 cells/µL (13%) lymphocytes (ref.

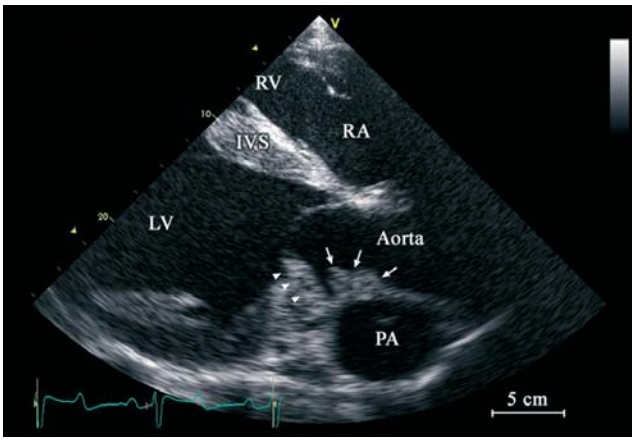

Figure 1. On cardiac ultrasound of the left ventricular outflow tract, a large vegetation is seen on the aortic valve cusps (arrow heads) and at the sinus of Valsalva (arrows). RA: right atrium; RV: right ventricle; IVS: interventricular septum; LV: left ventricle; PA: pulmonary artery. The displayed depth is 26 cm and dorsal is to the right of the image. The image is taken at end-diastole.

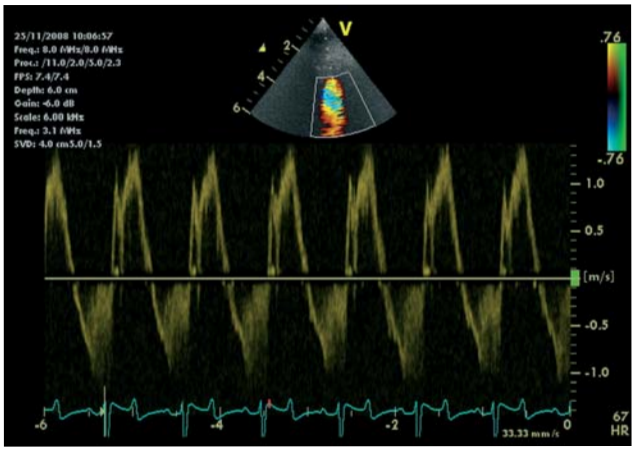

Figure 2. The spectral Doppler trace from the right carotid artery shows an increased systolic flow velocity of 131 cm/s. During diastole the flow is reversed with a peak velocity of -83 cm/s. The ECG is on the lower trace. The total duration of the clip is 6 seconds.

1500-7700 cells/µL) (Morris, 2009). Total protein was 67 g/L (ref. 58-77 g/L) (Carlson, 2009), with 17.3 g/L albumin (ref. 26-37 g/L) (Carlson, 2009) and 26.4 g/L gamma globulins (ref. 5.5-19 g/L) (Carlson, 2009). The packed cell volume (PCV) was 20% (ref. 32-48%) (Latimer and Rakich, 1992). The arterial partial carbon dioxide (p_aCO_2) and oxygen (p_aO_2) pressures were 33.5 mmHg (ref. 40.0-45.0 mmHg) and 93.4 mmHg (ref. > 86 mmHg) (Bayly *et al.*, 2002), respectively. A blood culture revealed Gram-negative hemolytic bacteria.

Cardiac troponin I (cTnI), measured with a luminescent immunoassay with a detection limit of 0.10 µg/L (ADVIA centaur®, Bayer Diagnostics), was 0.19 µg/L (ref. <0.15µg/L) (Begg *et al.*, 2006). The creatinine kinase's isoenzyme CK-MB was normal.

An electrocardiogram (ECG) showed sinus tachycardia at 64 bpm with occasional ventricular premature beats.

Pulmonary ultrasound showed comet tail artefacts all over the lung surface. On cardiac ultrasound (3S,

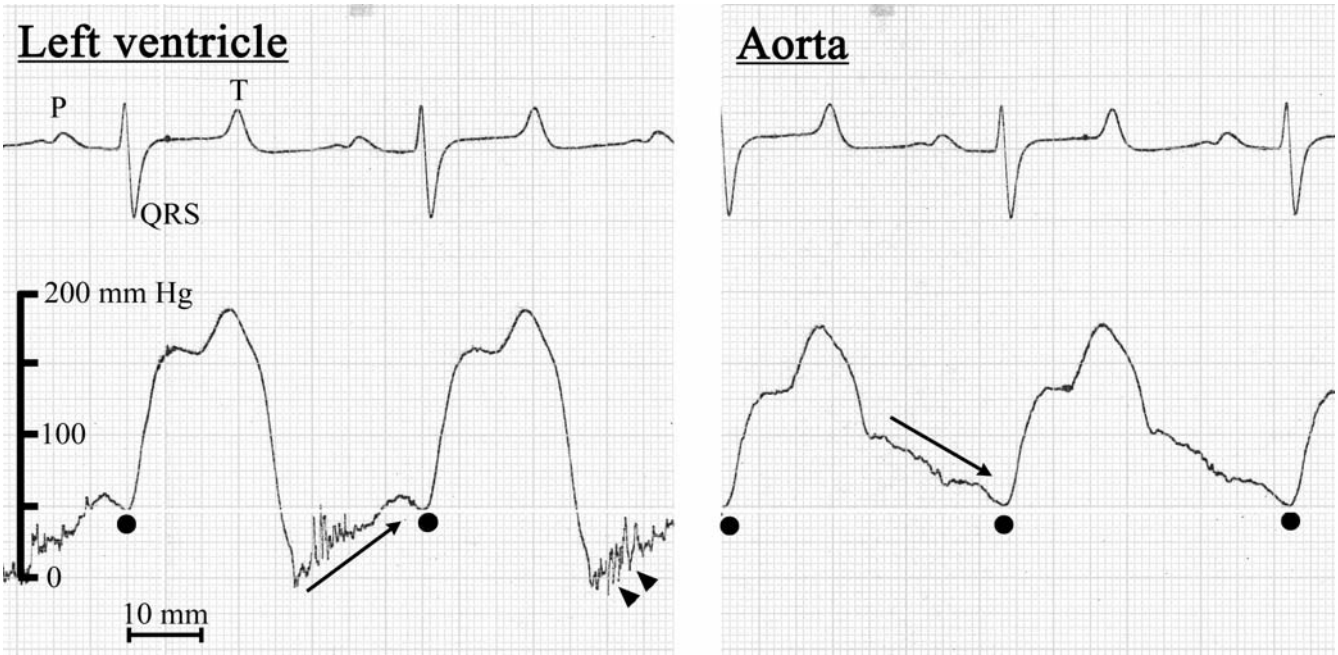

Figure 3. Simultaneous ECG (upper) and pressure (lower) recording of the left ventricle (left) and the aorta (right) at 50 mm/s. The systolic pressures in the left ventricle and the aorta are increased. The diastole is short because of the heart rate of 75/min. Nevertheless, during diastole, the left ventricular pressure increases by 50 mm Hg and the aortic pressure decreases by 50 mm Hg (arrows) because of the severe aortic regurgitation. At end-diastole (black dot), the left ventricular and aortic pressures are almost equal. A vibrating left ventricular signal is recorded in diastole due to impingement of the regurgitant jet and the pressure transducer (arrow heads).

Vivid7Pro, GE Healthcare), the left ventricle (LV) and the left atrium (LA) had a globoid aspect. The aortic valve was trileaflet. A 31x30 mm nodular thickening was present between the left and the right coronary cusp of the aortic valve (Figure 1). On the proximal aortic wall a vegetative lesion of about 19x32 mm was visible. On M-mode, a hyperkinetic movement of the interventricular septum was noted. On color flow Doppler severe aortic regurgitation (AoR) with prolapse of the aortic valve and mild mitral regurgitation were found.

The end-diastolic and systolic aortic diameters at the sino-tubular junction, measured from a right parasternal view, were 3.9 cm (ref. 6.8-8.4 cm) and 6.6 cm (ref. 6.6-8.5 cm), respectively (Patteson *et al.*, 1995a). The systolic and diastolic LV internal diameters at chordal level were 7.8 cm (ref. 5.8-8.8 cm) and 13.2 cm (ref. 9.7-13.1 cm) (Patteson *et al.*, 1995a), respectively. Fractional shortening (FS%) was 41% (ref. 29 – 44%) (Patteson *et al.*, 1995a). The LV pre-ejection period (PEP) was 0.032 s (ref. 0.076 ± 0.018 s), the LV ejection time (LVET) was 0.386 s (ref. 0.407 ± 0.03 s) and the PEP/LVET ratio was 0.08 (ref. 0.186 ± 0.04) (Patteson *et al.*, 1995b). Fluttering of the septal leaflet of the mitral valve was visualized on M-mode. The mitral valve E point to septal separation had a value of 0.5 cm (normal ≤ 1 cm) (Reef and Spencer, 1987). From a left parasternal view, the end-diastolic and systolic LA sizes were 14.4 cm (ref. 11.3-14.5 cm) and 14.3 cm (ref. 11.2-14.5 cm) (Patteson *et al.*, 1995a), respectively. The ratio between the LA diameter and the aortic diameter (LA/Ao) was 1.44 (ref. <1.2) (Young, 2004). The pulmonary artery diameter was 6.5 cm (ref. 5.2-6.9 cm) (Patteson *et al.*, 1995a).

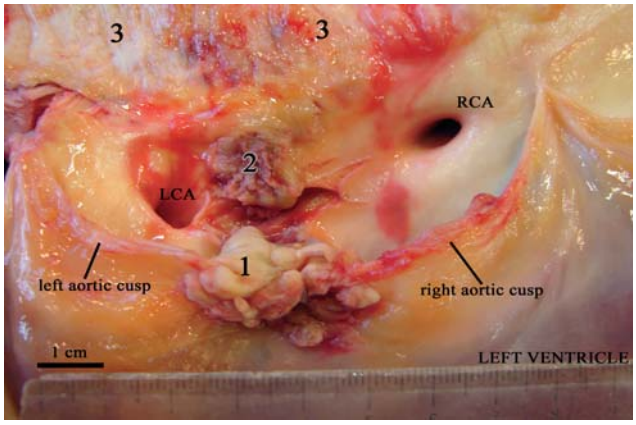

Figure 4. A large vegetation is present between the left and the right aortic cusps (1) and at the sinus of Valsalva (2), between the left and the right coronary arteries (LCA, RCA). An atherosclerotic plaque is present in the proximal aorta (3). Dorsal is to the top of the image.

With an 8 MHz transducer, a pulsed wave-Doppler recording of the right common carotid artery flow showed a systolic peak velocity of 131 cm/s (ref. 87.77 ± 10.68 cm/s) (Schmitz and Grabner, 2008) and a retrograde diastolic peak velocity of -83 cm/s (ref. -7.10 ± 7.18 cm/s) (Schmitz and Grabner, 2008), thus demonstrating a reverse flow during diastole (Figure 2).

A Millar Microtip catheter was inserted into the right common carotid artery to measure aortic and LV blood pressures. The LV end-diastolic pressure was 50 mmHg (ref. 11±2 mmHg) (Parks and Manohar, 1983) and the systolic pressure was 187 mmHg (reference 125±2 mmHg) (Rugh *et al.*, 1989) (Figure 3). A holodiastolic

vibration was present on the ventricular recording. The aortic end-diastolic pressure was also 50 mmHg (ref. 103±4 mmHg) (Parks and Manohar, 1983) and the systolic pressure was 175 mmHg (ref. 155±4 mmHg) (Parks and Manohar, 1983).

Because of the combination of severe aortic regurgitation and endocarditis, a grave prognosis was given. Further treatment was declined by the owner and euthanasia was elected.

POST MORTEM EXAMINATION

An irregular, opaque, whitish mass was present between the left and right aortic valve cusps (Figure 4). Near this mass, at the sinus of Valsalva, an additional vegetative mass of 2 cm in diameter was seen. The proximal aorta presented an atherosclerotic plaque (25 x 15 mm).

Samples of the aortic valve, lung, liver, kidney and synovium of the fetlocks were collected in 10% buffered formalin and embedded in paraffin wax, cut in 5µm-thick sections and stained with standard hematoxylin and eosin.

Histological examination revealed ulceration of the aortic valve cusp with an overlay of fibrin, admixed with cell debris and degenerated neutrophils. Multiple rod-shaped bacterial colonies were detected within the fibrinonecrotic mass. A diffuse infiltration of neutrophils was seen within the fibrosa and spongiosa.

Culture from the vegetative lesion revealed Gram-negative hemolytic bacteria, though the exact organism could not be identified.

Additional findings included diffuse passive congestion of the liver, lung edema, interstitial pneumonia, moderate interstitial nephritis and swollen fetlock joints due to chronic synovitis of the joint capsule involving increased amounts of synovial fluid with reduced viscosity.

DISCUSSION

AoR is frequently found in horses. Most commonly, it is caused by a degenerative ageing condition occurring in horses that are older than 10 years (Reef and Spencer, 1987; Marr, 1999). The exact mechanism is not fully understood, but it is probably related to a contractile dysfunction of the valve, together with the increasing collagen associated with increasing age (Bowen *et al.*, 2006). Bacterial endocarditis is a less common cause of AoR and it occurs more frequently in younger horses (Buergelt *et al.*, 1985; Reef and Spencer, 1987; Maxson and Reef, 1997; Porter *et al.*, 2008). Congenital AoR is rare, sometimes being associated with a ventricular septal defect (Reef and Spencer, 1987).

In the acute phase of AoR, the forward stroke volume will be decreased because the normal-sized LV is not able to compensate for the overfilling. On a pressure recording, the decrease in aortic pressure during diastole will be more rapid and more pronounced than normal. At the same time, the LV diastolic pressure will show a marked increase. In severe cases, both pressures can even equilibrate at end-diastole (Braunwald, 2001).

In the chronic phase, when AoR develops more slowly, the heart is able to generate a normal effective forward stroke volume and ejection fraction. This is accomplished by LV dilatation, which results in increased stroke volume. However, this will also result in LV systolic hypertension and increased wall tension. Consequently, eccentric hypertrophy, with fiber elongation and addition of sarcomeres in series, will occur in the heart's second attempt to compensate for the pressure overload. In compensated states of chronic AoR the myocardium is able to preserve a normal ratio between LV wall thickness and chamber radius, which leads to a normal end-diastolic wall tension, which in turn results in a normal effective ejection fraction (Rahimtoola, 2004; Otto and Bonow, 2008). Intra-ventricular and intra-aortic pressures at this stage of the disease will show a different trace compared to the acute situation. During diastole, the regurgitant flow from the aorta into the enlarged LV will cause a steep decrease of aortic pressure, even though the LV pressure will only slightly increase. Additionally, during systole, the increased stroke volume (forward stroke volume plus regurgitant volume) will result in higher aortic systolic pressures compared to the acute phase (Braunwald, 2001; Rahimtoola, 2004).

The pressure curves and ultrasound findings from this case point to a rather acute stage of the disease with limited myocardial remodeling. As the LV has a limited ability to dilate acutely, the diastolic pressure rise was huge and even equilibrated with the aortic pressure, which resulted in a decreased LV pre-ejection period. Because of this high diastolic pressure, pulmonary edema developed due to the increased left atrial and pulmonary pressures. In an attempt to improve cardiac output, the LV contractile function increases, as evidenced by the hyperkinetic septal motion, and compensatory tachycardia occurs. However, when these compensatory mechanisms also fail, more severe signs of heart failure occur, including increased pulmonary edema (Rahimtoola, 2004) and right-sided heart failure with ventral and distal limb edema. The large difference between the systolic and diastolic aortic diameters in this horse confirmed the hemodynamic importance of the aortic regurgitation. The LA/Ao ratio was increased due to the decreased aortic diameter in diastole. Because of the relatively acute stage of the disease, the pulmonary artery was still within the reference range, even though the widespread pulmonary comet tail artefacts indicated the presence of severe pulmonary edema.

In each case of AoR, it is important to palpate the peripheral arterial pulsation. In fact, the strength of the pulsation that one can feel represents the 'pulse pressure', which is the difference between the systolic and the diastolic arterial pressure. In the event of severe AoR, the pulsation can become strong and bounding, a condition that is called the 'Watson's water hammer pulse' or Corrigan's pulse. In this case, the water hammer pulse was so strong that it could be felt all over the body surface. The mechanism of this water hammer pulse in-

volves the wider pulse pressure due to the rapid upstroke with an increased systolic pressure (due to increased stroke volume, increased inotropy, etc.) and the rapid descent with the lower diastolic pressure (because of the run-off of the aorta and the rapid emptying of the arteries due to higher flow velocities) (Vakil *et al.*, 2001; Suvarna, 2008).

In the present case, the aortic pressure recording confirmed the wide pulse pressure. In addition, the carotid spectral Doppler trace also showed an increased systolic flow velocity with the reversal of flow during diastole, thus confirming this wide pulse pressure. Carotid reverse flow has been described in humans and in horses with severe aortic regurgitation (Cipone *et al.*, 1995; Schmitz and Grabner, 2008).

While compensatory mechanisms increase myocardial oxygen demand (myocardial remodeling, increased inotropy, shortened diastole, longer systole), coronary flow will decrease because of the decreased diastolic pressure in the aorta and the compression of the intramyocardial coronary arteries (Rahimtoola, 2004). This myocardial ischemia can result in cellular damage and arrhythmogenesis (Reimer *et al.*, 1992, Patteson, 1999). In addition, neuroendocrine changes, and especially the release of bacteria and emboli into the circulation leading to myocardial infarction and myocarditis, may induce cardiac arrhythmias (Maxson and Reef, 1997; Marr, 1999; Bonagura and Reef, 2004; Verdegaal and Sloet van Oldruitenborgh-Oosterbaan, 2006; Porter *et al.*, 2008). Both myocardial damage, resulting in an increased level of cTnI, and ventricular premature beats were found in this horse. The absence of an increase in CK-MB was probably related to the fact that this parameter is less sensitive than cTnI (Antman and Braunwald, 2008).

The treatment of bacterial endocarditis includes high doses of broad spectrum bactericidal antibiotics, preferably selected on the basis of the results of a blood culture. However, therapeutic antibiotic levels may be difficult to obtain due to the lack of blood penetration at the level of the vegetation. Therefore, drugs with good tissue penetration should be chosen. This is a long-term course of therapy and, in some cases, it can lead to a worsening of the regurgitation due to the healing of the vegetation and scaring of the valve. Before and throughout the treatment, it is necessary to keep in mind the bacterial isolate, the costs, the clinical evolution and the possible toxicosis that can be caused by the antimicrobial therapy. Although the prognosis for tricuspid endocarditis is guarded, most cases of bacterial endocarditis carry a poor prognosis, especially when diagnosed in an advanced stage of the disease (Maxson and Reef, 1997; Bonagura and Reef, 2004; Jesty and Reef, 2006; Verdegaal and Sloet van Oldruitenborgh-Oosterbaan, 2006). Even in cases of mild regurgitation, bacteriological cure is difficult to accomplish and healing of the vegetation may not solve the regurgitation and may even exacerbate it due to deformation of the valve. Therefore, a horse with endocarditis has a very low probability of becoming a performance or breeding animal again (Bonagura and Reef, 2004).

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De worm uit de staart snijden

Peet Van Damme van Sint-Laureins - God hebbe zijn ziele – werd tot vóór een paar tientallen jaren nog regelmatig door de boeren opgeroepen toen hun koeien "door de mare bereden werden". Hij stak zijn eigen groot scheermes in de zak en trok er op af zoals zijn grootvader en overgrootvader het hadden gedaan. Hij greep de staart van het zieke beest, schoor een bosje haar van het staarteind weg en sneed, precies waar hij moest snijden, een gleufje in de staart. Het beest stond recht, zwetend en kuchend, en was van de mare bevrijd.

Wat was de "mare"? Wat betekende het sneetje in de staart medisch gezien? Zoals Peet Van Damme leefden er nog wonderdokters, ook voor de mensen. Er waren er een hele hoop die komedie speelden en goud verdienden. Er waren er een hele hoop flagrante bedriegers onder, charlatans, bestweters, tovenaars, aflezers, zachtzinnige moordenaars.

Tot zover dit letterlijke citaat uit een artikel in 1960 (1 maart) gepubliceerd door de journalist Berten De Keyzer (B.D.K.) in het dagblad *De Gentenaar*. Dit sfeerstukje uit het Oost-Vlaamse polderdorp Sint-Laureins vermengt twee belangrijke thema's uit de volksdiergeneeskunde;: 'de worm' of 'de wolf uit de staart snijden' en 'door de mare bereden' worden. Dat is enigszins misleidend, want die twee thema's hebben nauwelijks iets met elkaar te maken.

Het sneetje onder de staart was bedoeld om een aanslepende, 'knagende' ziekteverwekker uit het lichaam te laten ontsnappen. Die noemde men, bij gebrek aan beter weten, 'de worm' of 'de wolf '. Men vindt dat geloof nog terug in uitdrukkingen als: 'de knagende worm van het geweten' en 'in cauda venenum': 'het venijn zit in de staart'. Dierenarts en volkskundige Jaak Wouters veronderstelt dat 'wolf' een vervorming is van 'worm'

Bij honden en zelfs katten deed men iets gelijkaardigs. Met een stopnaald rukte men 'de worm' weg van onder de tong. Dat heette dan 'de hond van de worm pellen'. Wouters geeft in zijn werk *Volksdiergeneeskunde* (1966) tal van voorbeelden uit diverse streken en uit de oude veterinaire literatuur. Bij dezelfde auteur gaan we ook te rade over de tweede component van het Peet Van Damme verhaal: 'van de mare bereden' (zie pg. ... of in een volgend nummer).

Luc Devriese